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Conclusions: Our results suggest that appropriate dose adjustments by reducing the intervals of IFX dosing may overcome low ADA titers with the aim to maintain or restore clinical response. In addition, proactive evaluation of ADA status may allow earlier identification of patients prone to a loss of response. The implementation of Bayesian dashboards may assist in the prediction of SICs <3 μg and determining adequate dosing modifications.

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Interstitial and granulomatous lung disease in inflammatory bowel disease patients

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Background: Granulomatous (GL) and interstitial lung disease (ILD) are rare respiratory disorders that have been associated with inflammatory bowel disease (IBD). Clinical presentation is polymorphic and aetiology is unclear.

Methods: This was European Crohn's and colitis organisation (ECCO) retrospective observational study performed as part of CONFER project. A call to all ECCO members was made to report concomitant granulomatous or ILD and IBD cases. Clinical data were recorded in a standardised case report form.

Results: Twenty-two granulomatous lung disease patients were identified from 18 university hospitals, 17 males and 5 females with a mean age of 46 years (18–86); 17 patients with Crohn's disease (CD) and five with ulcerative colitis (UC). In 19 patients IBD diagnosis preceded lung disease in a median time of 10.6 years (0–27). In three patients lung disease were diagnosed in a median time of 14 months (8–24) before IBD. Only four patients had active disease. Seven patients had drug-related granulomatous disease (sarcoidosis $n = 4$) and 14 had non-drug-related GL (primary sarcoidosis $n = 7$, fungal infection $n = 2$ and unspecified $n = 5$). Ten patients (45%) required hospitalisation but none required invasive ventilation. Fifteen of 22 patients received systemic steroids and causative drug was stopped in all patients. At further follow-up, 15 of 22 patients had no respiratory symptoms. Thirty-one patients with ILD were identified from 14 medical centres, 12 females and 19 males with mean age of 47 years (17–84); eight patients had CD, 22 had UC and one had indeterminate colitis. All patients had IBD diagnosis prior to ILD with a median time of 10.27 years (0.3–51). Eight patients had active disease. Eleven patients had non-drug-related ILD and 20 had drug-related ILD (mesalazine $n = 9$, methotrexate $n = 1$, golimumab $n = 1$, vedolizumab $n = 1$ and infliximab $n = 8$). ILD cases were classified as: Cryptogenic organising pneumonia $n = 11$, Eosinophilic pneumonitis $n = 2$, bronchiolitis $n = 2$, acute interstitial pneumonia $n = 8$, interstitial lung disease due to connective tissue $n = 1$, idiopathic fibrosis $n = 6$ and unclassified $n = 1$. Twenty-five patients (80.6%) required hospitalisation and one required non-invasive ventilation. Twenty-seven patients (87%) received systemic steroids and causative drug was stopped in all patients. At further follow-up, 14 of 31 patients had no respiratory symptoms, 7 of 31 had some improvement, and 4 of 31 had ongoing symptoms. There was one patient referred for consideration of lung transplant due to progressive fibrosis and one death due to mesalazine.

Conclusions: GL and ILD are rare, but require hospitalisation and systemic steroids. In our case series half of cases were drug-related but there was no signal of relationship between IBD therapy and the onset of ILD. More studies are needed to investigate the pathogenesis and causative association.

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Treatment naïve newly diagnosed patients with Crohn's disease have microbial dysbiosis correlated with disease activity and faecal calprotectin—results from a prospective inception cohort

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Background: Microbial dysbiosis is believed to play a role in Crohn's disease (CD). Most data are derived from CD patients under medications, an exposure that might impact microbial composition. Our aim was to assess microbial dysbiosis in patients with newly diagnosed CD and correlate it with disease activity.

Methods: Newly diagnosed CD patients were prospectively recruited and followed longitudinally. Clinical data, disease activity (physician global assessment [PGA] ranging from 0 to 3), and serum and faecal inflammatory biomarkers, were collected. Faecal samples were assessed for microbial composition using 16S rRNA sequencing. The microbial dysbiosis index (MDI) was used to quantify the degree of dysbiosis per sample.